EXHIBIT F

EXHIBIT H

UNITED STATES DISTRICT COURT
MIDDLE DISTRICT OF NORTH CAROLINA

JOAQUÍN CARCAÑO *et al.*, Plaintiffs, *v.* PATRICK MCCRORY *et al.*, Defendants	CASE NO. 1:16-CV-00236-TDS-JEP
UNITED STATES OF AMERICA, Plaintiff, *v.* STATE OF NORTH CAROLINA *et al.*, Defendants	CASE NO. 1:16-CV-00425-TDS-JEP

EXPERT DECLARATION OF Paul W Hruz, M.D., Ph.D

1. I have been retained by counsel for Defendants as an expert in connection with the above-captioned litigation. I have actual knowledge of the matters stated in this declaration. My professional background, experience, and publications are detailed in my curriculum vitae, a true and accurate copy which is attached as Exhibit A to this declaration. I received my doctor of philosophy degree from the Medical College of Wisconsin in 1993. I received my medical degree from the Medical College of Wisconsin in 1994. I am currently the Director of the Division of Pediatric Endocrinology and Diabetes at Washington University School of Medicine.

I served as the Director of the Pediatric Endocrinology Fellowship Program at Washington University from 2008-2016.

2. I am board certified in Pediatrics and Pediatric Endocrinology. I have been licensed to practice medicine in Missouri since 2000.

3. My professional memberships include the American Academy of Pediatrics, the Pediatric Endocrine Society, the Endocrine Society, and the American Association for Biochemistry and Molecular Biology.

4. I have extensive experience in treating infants and children with disorders of sexual development and am an active member of the multidisciplinary Disorders of Sexual Development (DSD) program at Washington University. The DSD Team at Washington University is part of the DSD-Translational Research Network, a national multi-institutional research network that investigates the genetic causes and the psychologic consequences of DSD.

5. In the nearly 20 years that I have been in clinical practice I have participated in the care of hundreds of children with disorders of sexual development including but not limited to congenital adrenal hyperplasia, 3β-hydroxysteroid dehydrogenase deficiency, partial and complete androgen insensitivity, 17-hydroxysteroid dehydrogenase deficiency, cloacal extrophy, aphallia, and Turner syndrome.

6. In my role as the director of the Division of Pediatric Endocrinology at Washington University, I have extensively studied the existing literature related to the incidence, potential etiology and treatment of gender dysphoria as efforts were made to develop a Transgender clinic at Saint Louis Children's Hospital. I have also participated in local and national meetings where the endocrine care of children with gender dysphoria has been discussed and debated. Pediatric patients referred to our practice for the evaluation and treatment of gender dysphoria are cared

for by an interdisciplinary team of providers that includes a psychologist and pediatric endocrinologist who have been specifically chosen for this role based upon a special interest in this rare patient population. Due to serious concerns regarding the safety, efficacy, and ethics of the current treatment paradigm, I have not directly engaged in hormonal treatment of patients with gender dysphoria.

7. My opinions as detailed in this declaration are based upon my knowledge and direct professional experience in the subject matters discussed. The materials that I have relied upon are the same types of materials that other experts in my field of clinical practice rely upon when forming opinions on the subject. A list of the sources I have relied on is attached as Exhibit B to this declaration.

8. Over my career, I have provided expert medical record review and testified at deposition in less than a dozen cases. I have never testified at trial and I have not been involved in any depositions in the past four years.

9. I am being compensated at an hourly rate for actual time devoted, at the rate of $350 per hour. My compensation does not depend on the outcome of this litigation, the opinions I express, or the testimony I provide.

Basic Terminology

10. Biological sex is a term that specifically refers to a member of a species in relation to the member's capacity to either donate (male) or receive (female) genetic material for the purpose of reproduction. This remains the standard definition that has been accepted and used by scientists, medical personnel, and society in general.

11. Gender, a term that had traditionally been reserved for grammatical purposes, is currently used to describe the psychologic and cultural characteristics of a person in relation to biological sex. Gender therefore exists in reference to societal perceptions, not biology.

12. Gender identity refers to a person's individual perception of being male or female.

13. Sexual orientation refers to a person's arousal and desire for sexual intimacy with members of the male or female sex.

Human sexuality in relation to fundamental biology and observed variations

14. Sex is genetically encoded at the moment of conception due to the presence of specific DNA sequences (i.e. genes) that direct the production of signals that influence the formation of the gonad to develop either into a testis or ovary. This genetic information is normally present on X and Y chromosomes. Chromosomal sex refers to the normal complement of X and Y chromosomes (i.e. normal human males have one X and one Y chromosome whereas normal human females have two X chromosomes). Genetic signals are mediated through the activation or deactivation of other genes and through programmed signaling of hormones and cellular transcription factors. The default pattern of development in the absence of external signaling is female. The development of the male appearance (phenotype) depends upon active signaling processes.

15. For members of the human species, sex is normatively aligned in a binary fashion (i.e., either male or female) in relation to biologic purpose. Medical designation of an individual as male or female is typically made at birth according to external phenotypic expression of primary sexual traits (i.e., presence of a penis for males and presence of labia and vagina for females).

16. Due to genetic and hormonal variation in the developing fetus, normative development of the external genitalia in any individual differs with respect to size and appearance while maintaining an ability to function with respect to biologic purpose (i.e. reproduction). Internal structures (e.g. gonad, uterus, vas deferens) normatively align with external genitalia.

17. Reliance upon external phenotypic expression of primary sexual traits is a highly accurate means to assign biologic sex. In over 99.9% of cases, this designation will correlate with internal sexual traits and capacity for normal biologic sexual function.

18. Due the complexity of signals that are involved in normal sexual development, it is not surprising that a small number of individuals are born with defects in this process. Defects can occur either through inherited or de novo mutations in genes that are involved in sexual determination or through environmental insults during critical states of sexual development. Persons who are born with such abnormalities are considered to have a disorder of sexual development (DSD). Most often, this is first detected as ambiguity in the appearance of the external genitalia.

19. Normal variation in external genital appearance (e.g. phallic size) does not alter the basic biologic nature of sex as a binary trait. "Intersex" conditions represent disorders of normal development, not a third sex.

20. Medical care of persons with DSDs is primarily directed toward identification of the etiology of the defect and treatment of any associated complications. Similar to other diseases, tools such as the Prader scale are used to stage the severity of the deviation from normal. In children with DSDs, characterization based upon phenotype alone does not reliably predict chromosomal sex nor does it necessarily correlate with potential for biological sexual function.

Decisions on initial sex assignment in these rare cases require detailed assessment by a team of expert medical providers.

21. Standard medical practice in the treatment of persons with DSDs has evolved with growing understanding of the physical and psychologic needs and outcomes for affected individuals. Previously, it was felt that a definitive sex assignment was necessary shortly after birth with the belief that this would allow patients with DSDs to best conform to the assigned sex. Current practice is to defer sex assignment until the etiology of the disorder is determined and, if possible, a prediction can be made on likely biologic and psychologic outcomes. When this cannot be done with confidence, a presumptive sex assignment is made. Factors used in making such decisions include chromosomal sex, phenotypic appearance of the external genitalia, and parental desires. The availability of new information can in rare circumstances lead to sex reassignment. Decisions on whether to surgically alter the external genitalia to align with sex are generally deferred until the patient is able to provide consent.

Gender Dysphoria in relation to Biological Sex

22. Although gender usually aligns with biological sex, some individuals experience discordance in these distinct traits. Specifically, biologic females may identify as males and biologic males may identify as females. As gender by definition is distinct from biological sex, one's gender identity does not change a person's biological sex.

23. Individuals who experience significant distress due to discordance between gender identity and sex are considered to have "gender dysphoria". Although the prevalence of gender dysphoria has not been established by rigorous scientific analysis, estimates reported in in the DSM-V are between 0.005% to 0.014% for adult males and 0.002% to 0.003% for adult females.

Thus, gender dysphoria is a rare condition. It is currently unknown whether these estimates are falsely low due to under-reporting, or if changing societal acceptance of transgenderism and the growing number of medical centers providing medical intervention for gender dysphoria affects the number of persons who identify as transgender. Recent data suggests that the number of people seeking care for gender dysphoria is increasing with some estimates as high as 4-fold.

24. Most people with gender dysphoria have normally formed and functional sexual organs. The etiology of gender dysphoria in these persons remains to be identified. Theories include prenatal hormone exposure, genetic variation, and postnatal environmental influences. Based upon the currently available but incomplete dataset, it is likely that gender dysphoria is multifactorial with differing qualitative and quantitative influences in any given individual. There is strong evidence against the theory that gender identity is determined at or before birth and is unchangeable. This comes from identical twin studies where siblings share genetic complements and prenatal environmental exposure but have differing gender identities.

25. Further evidence that gender identity is not fixed comes from well established peer reviewed literature demonstrating that the vast majority (80-95%) of children who express gender dysphoria revert to a gender identity concordant with their biological sex by late adolescence. It is not known whether individuals with gender dysphoria persistence have differing etiologies or severity of precipitating factors compared to desisting individuals.

26. The limited emerging data has suggested structural and functional differences between brains from normal and transgender individuals. These data do not establish whether these differences are innate and fixed or acquired and malleable. The remarkable neuronal plasticity of the brain is known and has been studied extensively in gender-independent contexts related to health and disease, learning and behavior.

Gender Ideology

27. The modern attempt to equate gender identity with sex is not based upon sound scientific principles but rather is based upon ideology fueled by advocacy. Although worldviews among scientists and physicians, similar to society at large, differ, science is firmly grounded in physical reality not perception. The inherent link between human sexual biology and teleology is self-evident and fixed.

28. The claims of proponents of transgenderism, which include opinions such as "Gender defines who one is at his/her core" and "Gender is the only true determinant of sex" must be viewed in their proper philosophical context. There is no scientific basis for redefining sex on the basis of a person's psychological sense of 'gender'. It is erroneous and potentially damaging to equate these opinions as established medical fact.

29. The prevailing, constant and accurate designation of sex as a biological trait grounded in the inherent purpose of male and female anatomy and as manifested in the appearance of external genitalia at birth remains the proper scientific and medical standard. Redefinition of what is normal based upon pathologic variation is not established medical fact.

Potential Harm Related to Gender Dysphoria Treatments

30. The fundamental purpose of the practice of medicine is to treat disease and alleviate suffering. An essential tenet of medical practice is to avoid doing harm in the process. Due to the frequent lack of clear and definitive evidence on how to best accomplish this goal, treatment approaches can and do frequently differ among highly knowledgeable, competent, and caring physicians.

31. Persons with gender dysphoria as delineated in the DSM-V experience significant psychological distress related to their condition with elevated risk of depression, suicide, and other morbidities. Thus, attempts to provide effective medical care to affected persons are clearly warranted.

32. Efforts to effectively treat persons with gender dysphoria require respect for the inherent dignity of those affected, sensitivity to their suffering, and maintenance of objectivity in assessing etiologies and long-term outcomes. Desistance (i.e. reversion to gender identity concordant with sex) provides the greatest lifelong benefit and is the outcome in the majority of patients and should be maintained as a desired goal. Any intervention that interferes with the likelihood of resolution is unwarranted and potentially harmful.

33. There is an urgent need for high quality controlled clinical research trials to determine ways to develop supportive dignity affirming social environments that maintain affirmation of biological reality.

34. The Endocrine Society published in 2009 clinical guidelines for the treatment of gender dysphoric patients which include temporary suppression of pubertal development of children with GnRH agonists (hormone blockers normally used for children experiencing precocious puberty) followed by hormonal treatments to induce the development of secondary sexual traits consistent with one's gender identity. This guideline was developed using the GRADE (Recommendations, Assessment, Development, and Evaluation) system for rating clinical guidelines. As directly stated in the Endocrine Society publication, "the strength of recommendations and the quality of evidence was low or very low." According to the GRADE system, low recommendations indicate "Further research is very likely to have an important

impact on our confidence in the estimate of effect and is likely to change the estimate". Very low recommendations mean that "any estimate of effect is very uncertain".

35. There is little or no data to support pubertal suppression as a safe or effective treatment for gender dysphoria in children or adolescents. As noted, it is well established that 80-95% of children with gender dysphoria will resolve by the end of puberty without direct intervention to affirm transgender identity. Unfavorable long-term psychiatric outcomes for transgender adults point to gender resolution following puberty as the best hope for gender dysphoric children and adolescents.

36. In addition, treatment of gender dysphoric children with hormonal treatment (pubertal suppression and cross-hormone therapy) carries significant risk. It is generally accepted, even by advocates of transgender hormone therapy, that hormonal treatment results in sterility which in many cases is irreversible. Emerging data also show that treated patients have lower bone density which may lead to increased fracture risk later in life. Other potential adverse effects include disfiguring acne, high blood pressure, weight gain, abnormal glucose tolerance, breast cancer, liver disease, thrombosis, and cardiovascular disease.

37. Since strategies for the treatment of transgendered children as summarized by the Endocrine Society guidelines are relatively new, long-term outcomes are unknown. Evidence presented as support for short term reductions in psychological distress following social transition in a "gender affirming" environment remains inconclusive. When considered apart from advocacy based agendas, multiple potential confounders are evident. The most extensive long-term data on this question comes from the Dutch experience. Although appropriate caution is warranted in extrapolating these outcomes with current treatments, adults who have undergone

social transition with or without surgical modification of external genitalia continue to have rates of depression and suicide far above the background population.

38. With regard to public restrooms and other intimate facilities, there is no evidence to support social measures that promote or encourage gender transition as a medically necessary or effective treatment for gender dysphoria. If anything, one might expect that such social affirmation measures would interfere with known rates of gender resolution. Any activity that encourages or perpetuates transgender persistence for those who would otherwise desist can cause significant harm, including permanent sterility, to these persons. This is particularly concerning given that children are likely incapable of making informed consent to castrating treatments.

39. There remains a significant and unmet need to better understand both the biological, psychological, and environmental basis for the manifestation of discordance of gender identity in affected individuals together with rigorous controlled investigation of long-term outcomes including adverse consequences of attempted intervention. Uncontrolled social experimentation including the forced acceptance of altered norms for distinguishing persons according to biological sex is a potentially harmful and unscientific approach to dealing with this serious condition.

Pursuant to 28 U.S.C § 1746, I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Date: 08/09/2016

Signed: Paul W Hruz

Paul W. Hruz, M.D., Ph.D

Exhibit B

Hruz Sources

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69. VanderLaan, D. P., Leef, J. H., Wood, H., Hughes, S. K., and Zucker, K. J. (2015) Autism spectrum disorder risk factors and autistic traits in gender dysphoric children. *J Autism Dev Disord* **45**, 1742-1750]
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71. Wallien, M. S., and Cohen-Kettenis, P. T. (2008) Psychosexual outcome of gender-dysphoric children. *J Am Acad Child Adolesc Psychiatry* **47**, 1413-1423
72. Williams, Robert Hardin., and P. Reed. Larsen. *Williams Textbook of Endocrinology*. Philadelphia, PA: Saunders, 2003. Print.
73. Wood, H., Sasaki, S., Bradley, S. J., Singh, D., Fantus, S., Owen-Anderson, A., Di Giacomo, A., Bain, J., and Zucker, K. J. (2013) Patterns of referral to a gender identity service for children and adolescents (1976-2011): age, sex ratio, and sexual orientation. *J Sex Marital Ther* **39**, 1-6
74. World Health Organization, International Classification of Diseases, 10th edition
75. Yang, S., Cranford, J. A., Li, R., Zucker, R. A., and Buu, A. (2015) A time-varying effect model for studying gender differences in health behavior. *Stat Methods Med Res*
76. Zucker, K. J. (1999) Gender identity disorder in the DSM-IV. *J Sex Marital Ther* **25**, 5-9
77. Zucker, K. J. (2002) Evaluation of sex- and gender-assignment decisions in patients with physical intersex conditions: a methodological and statistical note. *J Sex Marital Ther* **28**, 269-274

78. Zucker, K. J. (2002) Intersexuality and gender identity differentiation. *J Pediatr Adolesc Gynecol* **15**, 3-13
79. Zucker, K. J. (2004) Gender identity development and issues. *Child Adolesc Psychiatr Clin N Am* **13**, 551-568, vii
80. Zucker, K. J. (2005) Gender identity disorder in children and adolescents. *Annu Rev Clin Psychol* **1**, 467-492
81. Zucker, K. J. (2008) On the "natural history" of gender identity disorder in children. *J Am Acad Child Adolesc Psychiatry* **47**, 1361-1363
82. Zucker, K. J. (2010) The DSM diagnostic criteria for gender identity disorder in children. *Arch Sex Behav* **39**, 477-498
83. Zucker, K. J. (2010) Reports from the DSM-V Work Group on sexual and gender identity disorders. *Arch Sex Behav* **39**, 217-220
84. Zucker, K. J., Beaulieu, N., Bradley, S. J., Grimshaw, G. M., and Wilcox, A. (2001) Handedness in boys with gender identity disorder. *J Child Psychol Psychiatry* **42**, 767-776
85. Zucker, K. J., Bradley, S. J., Ben-Dat, D. N., Ho, C., Johnson, L., and Owen, A. (2003) Psychopathology in the parents of boys with gender identity disorder. *J Am Acad Child Adolesc Psychiatry* **42**, 2-4
86. Zucker, K. J., Bradley, S. J., Doering, R. W., and Lozinski, J. A. (1985) Sex-typed behavior in cross-gender-identified children: stability and change at a one-year follow-up. *J Am Acad Child Psychiatry* **24**, 710-719
87. Zucker, K. J., Bradley, S. J., and Hughes, H. E. (1987) Gender dysphoria in a child with true hermaphroditism. *Can J Psychiatry* **32**, 602-609
88. Zucker, K. J., Bradley, S. J., Kuksis, M., Pecore, K., Birkenfeld-Adams, A., Doering, R. W., Mitchell, J. N., and Wild, J. (1999) Gender constancy judgments in children with gender identity disorder: evidence for a developmental lag. *Arch Sex Behav* **28**, 475-502
89. Zucker, K. J., Bradley, S. J., Owen-Anderson, A., Kibblewhite, S. J., and Cantor, J. M. (2008) Is gender identity disorder in adolescents coming out of the closet? *J Sex Marital Ther* **34**, 287-290
90. Zucker, K. J., Bradley, S. J., Owen-Anderson, A., Kibblewhite, S. J., Wood, H., Singh, D., and Choi, K. (2012) Demographics, behavior problems, and psychosexual characteristics of adolescents with gender identity disorder or transvestic fetishism. *J Sex Marital Ther* **38**, 151-189
91. Zucker, K. J., Bradley, S. J., and Sanikhani, M. (1997) Sex differences in referral rates of children with gender identity disorder: some hypotheses. *J Abnorm Child Psychol* **25**, 217-227
92. Zucker, K. J., Bradley, S. J., Sullivan, C. B., Kuksis, M., Birkenfeld-Adams, A., and Mitchell, J. N. (1993) A gender identity interview for children. *J Pers Assess* **61**, 443-456
93. Zucker, K. J., Finegan, J. K., Doering, R. W., and Bradley, S. J. (1984) Two subgroups of gender-problem children. *Arch Sex Behav* **13**, 27-39
94. Zucker, K. J., Green, R., Coates, S., Zuger, B., Cohen-Kettenis, P. T., Zecca, G. M., Lertora, V., Money, J., Hahn-Burke, S., Bradley, S. J., and Blanchard, R. (1997) Sibling sex ratio of boys with gender identity disorder. *J Child Psychol Psychiatry* **38**, 543-551
95. Zucker, K. J., Green, R., Garofano, C., Bradley, S. J., Williams, K., Rebach, H. M., and Sullivan, C. B. (1994) Prenatal gender preference of mothers of feminine and masculine boys: relation to sibling sex composition and birth order. *J Abnorm Child Psychol* **22**, 1-13
96. Zucker, K. J., Lawrence, A. A., and Kreukels, B. P. (2016) Gender Dysphoria in Adults. *Annu Rev Clin Psychol* **12**, 217-247
97. Zucker, K. J., and Wood, H. (2011) Assessment of gender variance in children. *Child Adolesc Psychiatr Clin N Am* **20**, 665-680
98. Zucker, K. J., Wood, H., Wasserman, L., VanderLaan, D. P., and Aitken, M. (2016) Increasing Referrals for Gender Dysphoria. *J Adolesc Health* **58**, 693-694

Curriculum Vitae

Paul W. Hruz, MD, PhD

Date: August 9, 2016

Personal Information

Date of birth: November 22, 1965
Place of birth: WI
Citizenship: USA

Address and Telephone Numbers

University: Washington University School of Medicine
Department of Pediatrics
Division of Endocrinology and Diabetes
660 South Euclid Avenue, Campus Box 8208
St. Louis, MO 63110
Phone: 314-286-2797
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email: hruz_p@kids.wustl.edu

Present Position

Associate Professor of Cell Biology and Physiology
Associate Professor of Pediatrics
Division Director, Pediatric Endocrinology and Diabetes

Education and Training

1987	B.S., Chemistry, Marquette University, Milwaukee, WI
1993	Ph.D., Biology and Physiology, Medical College of Wisconsin, Milwaukee, WI
1994	M.D., Medicine, Medical College of Wisconsin, Milwaukee, WI
1994 - 1997	Pediatric Residency, University of Washington - Pediatric , Seattle, Washington
1997 - 2000	Pediatric Endocrinology Fellowship, Washington University - Pediatric Endocrinology , Saint Louis, MO

Academic Positions and Employment

1996 - 1997	Locum Tenens Physician, Group Health of Puget Sound Eastside Hospital, Group Health of Puget Sound Eastside Hospital, Seattle , WA
2000 - 2003	Instructor of Pediatrics, Washington University, St. Louis, MO
2003 - 2011	Assistant Professor of Pediatrics, Washington University, St. Louis, MO

2004 - 2011 Assistant Professor of Cell Biology and Physiology, Washington University, St. Louis, MO
2011 - Pres Associate Professor of Pediatrics, Washington University, St. Louis, MO
2011 - Pres Associate Professor of Cell Biology and Physiology, Washington University, St. Louis, MO
2012 - Pres Division Director, Pediatric Endocrinology and Diabetes, Washington University, St. Louis, MO

Appointments and Committees

NIH Study Sections:
2005 NIH- NIDDK Special Emphasis Panel ZDK1 GRB-6 (Non-Standing Member)
2009 NIH- ACE Competitive Revisions ZRG1 AARR-H (95) S (Non-Standing Member)
2009 NIH- AIDS and AIDS Related Research IRG (Standing Member)
2011 NIH- Pediatric Endocrinologist K12 ZDK1 GRB-C (Non-Standing Member)
2014 NIH- Special Emphasis Panel ZRG1 BBBPY 58 (Non-Standing Member)
2014 NIH- AIDS and AIDS Related Research IRG (Standing Member)
2015 NIH- Cardiovascular and Respiratory Sciences Special Emphasis Panel ZDK1 GRB-J (02) (Non-Standing Member)
2015 NIH- NIDDK Special Emphasis Panel ZRG1 CVRS-Q (80) (Non-Standing Member)

University Affiliations:
2008 - Pres Director, Pediatric Endocrinology & Diabetes Fellowship Program
2010 - Pres Pediatric Computing Facility Advisory Committee
2012 - Pres Disorders of Sexual Development Interdisciplinary Care Program
2012 - Pres Director, Division of Pediatric Endocrinology & Diabetes
2014 - Pres Research Consultant, ICTS Research Forum - Child Health
2014 - Pres Director, Pediatric Diabetes Research Consortium

Hospital Affiliations:
2000 - Pres Attending Physician, St. Louis Children's Hospital

Thesis Committees (* Chair)		Advisor
2008 - 2011	Kelly Diggs-Andrews	Simon Fisher
2008 - 2010	Irwin Puentes	Simon Fisher
2008 - 2010	Tony Frovola	Kelle Moley
2009 - 2010	Lauren Flessner	Kelle Moley
2010 - 2012	Katie Boehle	Kelle Moley
2010 - 2013	Candace Reno*	Simon Fisher
2011 -Pres	Thomas Kraft	Paul Hruz
2013 - 2015	Chi Lun Pui	Audrey Odom
2013 -Pres	Leah Imlay	Audrey Odom
2014 -Pres	Anne Robinson	Katie Henzler-Wildman

2015 -Pres Allyson Mayer Brian DeBosch

Scholarship Oversight Committees
2013 -Pres Brittany Knipsein (Advisor: David Rudnick)

Licensure and Certifications

1997 - 2016 Board Certified in General Pediatrics
2000 - 2014 MO State License #2000155004
2001 - Pres Board Certified in Pediatric Endocrinology & Metabolism

Honors and Awards

1987 National Institute of Chemists Research and Recognition Award
1987 Phi Beta Kappa
1987 Phi Lambda Upsilon (Honorary Chemical Society)
1988 American Heart Association Predoctoral Fellowship Award
1994 Alpha Omega Alpha
1994 Armond J. Quick Award for Excellence in Biochemistry
1994 NIDDK/Diabetes Branch Most Outstanding Resident
1998 Pfizer Postdoctoral Fellowship Award
2002 Scholar, Child Health Research Center of Excellence in Developmental Biology at Washington University
2013 Julio V Santiago, M.D. Scholar in Pediatrics

Editorial Responsibilities

Editorial Boards:
2014 - Pres Endocrinology and Metabolism Clinics of North America

Ad Hoc Reviewer:
AIDS
AIDS Research and Human Retroviruses
American Journal of Pathology
American Journal of Physiology
British Journal of Pharmacology
Circulation Research
Clinical Pharmacology & Therapeutics
Comparative Biochemistry and Physiology
Diabetes
Experimental Biology and Medicine
Future Virology
Journal of Antimicrobial Chemotherapy
Journal of Biological Chemistry
Journal of Clinical Endocrinology & Metabolism
Journal of Molecular and Cellular Cardiology
Obesity Research

Professional Societies and Organizations

1992 - 2004	American Medical Association
1994 - 2005	American Academy of Pediatrics
1995 - 2014	American Association for the Advancement of Science
1998 - Pres	American Diabetes Association
1998 - Pres	Endocrine Society
1999 - Pres	Pediatric Endocrine Society
2004 - Pres	American Society for Biochemistry and Molecular Biology
2004 - Pres	Society for Pediatric Research
2004 - 2007	American Chemical Society
2005 - Pres	Full Fellow of the American Academy of Pediatrics
2013 - Pres	International Society for Pediatric and Adolescent Diabetes

Major Invited Professorships and Lectures

2002	St. Louis Children's Hospital, Pediatric Grand Rounds, St. Louis, MO
2004	National Disease Research Interchange, Human Islet Cell Research Conference, Philadelphia, PA
2004	NIDA-NIH Sponsored National Meeting on Hormones, Drug Abuse and Infections, Bethesda, MD
2005	The Collaborative Institute of Virology, Complications Committee Meeting, Boston, MA
2005	University of Indiana, Endocrine Grand Rounds, Indianapolis, IN
2006	Metabolic Syndrome Advisory Board Meeting, Bristol-Meyers Squibb, Pennington, NJ
2007	American Heart Association and American Academy of HIV Medicine State of the Science Conference: Initiative to Decrease Cardiovascular Risk and Increase Quality of Care for Patients Living with HIV/AIDS, Chicago, IL
2007	Medical College of Wisconsin, MSTP Annual Visiting Alumnus Lecture, Milwaukee, WI
2007	St Louis Children's Hospital, Pediatric Grand Rounds, St Louis, MO
2007	University of Arizona, Minority Access to Research Careers Seminar, Tucson AZ
2008	Boston University, Division of Endocrinology, Diabetes and Nutrition, Boston, MA
2009	St Louis Children's Hospital, Pediatric Grand Rounds, St Louis, MO
2010	American Diabetes Association Scientific Sessions, Symposium Lecture Orlando, FL
2010	University of Missouri Kansas City, School of Biological Sciences, Kansas City, MO
2011	Life Cycle Management Advisory Board Meeting, Bristol-Myers Squibb, Chicago, IL
2013	St Louis Children's Hospital, Pediatric Grand Rounds, St Louis MO
2013	St Louis Children's Hospital CPU Lecture, St Louis MO

2014 Pediatric Academic Societies Meeting, Vancouver, Canada, May 5, 2014
2014 American Diabetes Association 74th Scientific Sessions, San Francisco, CA, June 13, 2014

Consulting Relationships and Board Memberships

1996 - 2012 Consultant, Bristol Myers Squibb
1997 - 2012 Consultant, Gilead Sciences

Research Support

Governmental Support

R01 (Hruz) 9/20/2009 - 5/31/2014 (NCE)
NIH
Direct Effects of Antiretroviral Therapy on Cardiac Energy Homeostasis
The goal of this project is to characterize the influence of antiretroviral therapies on myocardial energy homeostasis and to elucidate how these changes in substrate delivery adversely affect cardiac function in the stressed heart.
Role: Principal Investigator

R01 (Hruz) 4/1/2007 - 1/31/2012 (NCE)
NIH
Mechanisms for Altered Glucose Homeostasis During HAART
The goal of this project is to identify the cellular targets of HIV protease inhibitors that lead to peripheral insulin resistance, impaired beta-cell function, and alterations in hepatic glucose production and to elucidate the molecular mechanisms of these effects.
Role: Principal Investigator

Non-Governmental Support

Research Program (Hruz) 6/1/2009 - 5/31/2012 (NCE)
MOD
Regulation of GLUT4 Intrinsic Activity
The major goals of this project are to investigate the ability of the GLUT4 tethering protein TUG and an UBL-domain containing N-terminal fragment of this protein to alter the intrinsic activity of the insulin responsive facilitative glucose transporter, to determine whether protein ubiquitination influences this association, and to characterize the role of the GLUT4 binding site on the modulation of glucose transport.
Role: Principal Investigator

(Hruz) 3/9/2010 - 6/8/2011 (NCE)
Bristol-Myers Squibb
Protective Effect of Saxagliptin on a Progressive Deterioration of Cardiovascular Function
Role: Principal Investigator

(Hruz)
Gilead Pharma

Novel HIV Protease Inhibitors and GLUT4
Role: Principal Investigator

II (Hruz) 2/1/2008 - 1/31/2011 (NCE)
CDI
Insulin Resistance and Myocardial Glucose Metabolism in Pediatric Heart Failure
Role: Co-Principal Investigator

Completed Support
R01 Student Supp (Hruz) 6/10/2009 - 8/31/2011
NIH
Mechanisms for Altered Glucose Homeostasis During HAART

II (Hruz) 2/1/2012 - 1/31/2015
CDI
Solution-State NMR Structure and Dynamics of Facilitative Glucose Transport Proteins

Past Trainees

2002 - 2002 Nishant Raj- Undergraduate Student (Other)
Study area: Research

2003 - 2004 Johann Hertel (Medical Student)
Study area: Research
Present position: Assistant Professor, University of North Carolina, Chapel Hill, NC

2003 John Paul Shen (Medical Student)
Study area: Research

2004 - 2005 Carl Cassel- High School Student (Other)
Study area: Research

2004 - 2004 Christopher Hawkins- Undergraduate Student (Other)
Study area: Research

2004 - 2004 Kaiming Wu- High School Student (Other)
Study area: Research

2005 Helena Johnson (Graduate Student)

2005 Jeremy Etzkorn (Medical Student)
Study area: Research
Present position: Assistant Professor, University of Pennsylvania

2006 Ramon Jin (Graduate Student)
Study area: Research

2006 Taekyung Kim (Graduate Student)
Study area: Research

2007 - 2008 Kai-Chien Yang (Graduate Student)
Study area: Research
Present position: Postdoctoral Research Associate, University of Chicago

2007 Paul Buske (Graduate Student)
Study area: Research
Present position: Postdoctoral Fellow, UCSF, San Francisco CA

2007	Randy Colvin (Medical Student) Study area: Research
2007 - 2007	Jan Freiss- Undergraduate Student (Other) Study area: Research
2008 - 2011	Arpita Vyas, MD (Clinical Fellow) Study area: Research Present position: Assistant Professor, Michigan State University, Lansing MI
2008 - 2009	Candace Reno (Graduate Student) Study area: Research Present position: Research Associate, University of Utah
2008	Temitope Aiyejorun (Grad Student) Study area: Research
2008 - 2012	Dennis Woo- Undergraduate Student (Other) Study area: Research Present position: MSTP Student, USC, Los Angeles CA
2009	Stephanie Scherer (Grad Student) Study area: Research
2009	Anne-Sophie Stolle- Undergraduate Student (Other) Study area: Research
2009 - 2009	Matthew Hruz- High School Student (Other) Study area: Research Present position: Computer Programmer, Consumer Affairs, Tulsa OK
2010	Constance Haufe- Undergraduate Student (Other) Study area: Research
2010 - 2011	Corinna Wilde- Undergraduate Student (Other) Study area: Researcher
2010 - 2010	Samuel Lite- High School Student (Other) Study area: Research
2011 - 2011	Amanda Koenig- High School Student (Other) Study area: Research
2011 - 2012	Lisa Becker- Undergraduate Student (Other)
2011 - 2011	Melissa Al-Jaoude- High School Students (Other)
2002 - 2010	Joseph Koster, PhD (Postdoc Fellow) Study area: Research
2005	Dominic Doran, DSc (Postdoctoral Fellow) Study area: HIV Protease Inhibitor Effects on Exercise Tolerance Present position: Faculty of Science, Liverpool John Moores Institute
2014 - 2014	David Hannibal (Clinical Research Trainee)
2010 - 2014	Lauren Flessner, PhD (Postdoctoral Fellow) Present position: Instructor, Syracuse University
2011 - 2016	Thomas Kraft (Graduate Student) Study Area: Glucose transporter structure/function Present position: Postdoctoral Fellow, Roche, Penzberg, Germany

Clinical Responsibilities

General Pediatrician, General Pediatric Ward Attending: 2-4 weeks per year, St. Louis Children's Hospital
Pediatric Endocrinologist, Endocrinology Night Telephone Consult Service: Average of 2-6 weeks/per year, St. Louis Children's Hospital
Pediatric Endocrinologist, Inpatient Endocrinology Consult Service: 4-6 weeks per year, St. Louis Children's Hospital
Pediatric Endocrinologist, Outpatient Endocrinology Clinic: Approximately 50 patient visits per month, St. Louis Children's Hospital

Teaching Responsibilities

Facilitator, Biology 5011- Ethics and Research Science, 6 hours/year
Facilitator, Cell Biology Graduate Student Journal Club, 4 hour/year
Facilitator, Discussion: Pituitary, Growth & Gonadal Cases, 2 hours/year
Facilitator, Medical Student Endocrinology and Metabolism Course, Small group
Lecturer, Cell Signaling Course, Diabetes module, 3 hours/year
Lecturer, Markey Course-Diabetes Module
Lecturer, Medical Student Growth Lecture (Women and Children's Health Rotation): Variable
Lecturer, Metabolism Clinical Rounds/Research Seminar: Presentations twice yearly
Lecturer, Pediatric Endocrinology Journal Club: Presentations yearly

Publications

1. Hruz, P. W., Narasimhan, C., Miziorko, H. M. (1992). 3-Hydroxy-3-methylglutaryl coenzyme A lyase: affinity labeling of the Pseudomonas mevalonii enzyme and assignment of cysteine-237 to the active site. *Biochemistry*, 31 (29), 6842-7 PubMed: 1637819.
2. Hruz, P. W., Miziorko, H. M. (1992). Avian 3-hydroxy-3-methylglutaryl-CoA lyase: sensitivity of enzyme activity to thiol/disulfide exchange and identification of proximal reactive cysteines. *Protein Sci*, 1 (9), 1144-53. PMCID: PMC2142181 PubMed: 1304393.
3. Mitchell, G. A., Robert, M. F., Hruz, P. W., Wang, S., Fontaine, G., Behnke, C. E., Mende-Mueller, L. M., Schappert, K., Lee, C., Gibson, K. M., Miziorko, H. M. (1993). 3-Hydroxy-3-methylglutaryl coenzyme A lyase (HL). Cloning of human and chicken liver HL cDNAs and characterization of a mutation causing human HL deficiency. *J Biol Chem*, 268 (6), 4376-81 PubMed: 8440722.
4. Hruz, P. W., Anderson, V. E., Miziorko, H. M. (1993). 3-Hydroxy-3-methylglutaryldithio-CoA: utility of an alternative substrate in elucidation of a role for HMG-CoA lyase's cation activator. *Biochim Biophys Acta*, 1162 (1-2), 149-54 PubMed: 8095409.
5. Roberts, J. R., Narasimhan, C., Hruz, P. W., Mitchell, G. A., Miziorko, H. M. (1994). 3-Hydroxy-3-methylglutaryl-CoA lyase: expression and isolation of the recombinant

human enzyme and investigation of a mechanism for regulation of enzyme activity. *J Biol Chem*, 269 (27), 17841-6 PubMed: 8027038.
6. Hruz, P. W., Mueckler, M. M. (1999). Cysteine-scanning mutagenesis of transmembrane segment 7 of the GLUT1 glucose transporter. *J Biol Chem*, 274 (51), 36176-80 PubMed: 10593902.
7. Murata, H., Hruz, P. W., Mueckler, M. (2000). The mechanism of insulin resistance caused by HIV protease inhibitor therapy. *J Biol Chem*, 275 (27), 20251-4 PubMed: 10806189.
8. Hruz, P. W., Mueckler, M. M. (2000). Cysteine-scanning mutagenesis of transmembrane segment 11 of the GLUT1 facilitative glucose transporter. *Biochemistry*, 39 (31), 9367-72 PubMed: 10924131.
9. Hruz, P. W., Mueckler, M. M. (2001). Structural analysis of the GLUT1 facilitative glucose transporter (review). *Mol Membr Biol*, 18 (3), 183-93 PubMed: 11681785.
10. Hruz, P. W., Murata, H., Mueckler, M. (2001). Adverse metabolic consequences of HIV protease inhibitor therapy: the search for a central mechanism. *Am J Physiol Endocrinol Metab*, 280 (4), E549-53 PubMed: 11254460.
11. Murata, H., Hruz, P. W., Mueckler, M. (2002). Investigating the cellular targets of HIV protease inhibitors: implications for metabolic disorders and improvements in drug therapy. *Curr Drug Targets Infect Disord*, 2 (1), 1-8 PubMed: 12462148.
12. Hruz, P. W., Murata, H., Qiu, H., Mueckler, M. (2002). Indinavir induces acute and reversible peripheral insulin resistance in rats. *Diabetes*, 51 (4), 937-42 PubMed: 11916910.
13. Murata, H., Hruz, P. W., Mueckler, M. (2002). Indinavir inhibits the glucose transporter isoform Glut4 at physiologic concentrations. *AIDS*, 16 (6), 859-63 PubMed: 11919487.
14. Koster, J. C., Remedi, M. S., Qiu, H., Nichols, C. G., Hruz, P. W. (2003). HIV protease inhibitors acutely impair glucose-stimulated insulin release. *Diabetes*, 52 (7), 1695-700. PMCID: PMC1403824 PubMed: 12829635.
15. Liao, Y., Shikapwashya, O. N., Shteyer, E., Dieckgraefe, B. K., Hruz, P. W., Rudnick, D. A. (2004). Delayed hepatocellular mitotic progression and impaired liver regeneration in early growth response-1-deficient mice. *J Biol Chem*, 279 (41), 43107-16 PubMed: 15265859.
16. Shteyer, E., Liao, Y., Muglia, L. J., Hruz, P. W., Rudnick, D. A. (2004). Disruption of hepatic adipogenesis is associated with impaired liver regeneration in mice. *Hepatology*, 40 (6), 1322-32 PubMed: 15565660.
17. Hertel, J., Struthers, H., Horj, C. B., Hruz, P. W. (2004). A structural basis for the acute effects of HIV protease inhibitors on GLUT4 intrinsic activity. *J Biol Chem*, 279 (53), 55147-52. PMCID: PMC1403823 PubMed: 15496402.
18. Yan, Q., Hruz, P. W. (2005). Direct comparison of the acute in vivo effects of HIV protease inhibitors on peripheral glucose disposal. *J Acquir Immune Defic Syndr*, 40 (4), 398-403. PMCID: PMC1360159 PubMed: 16280693.
19. Hruz, P. W. (2006). Molecular Mechanisms for Altered Glucose Homeostasis in HIV Infection. *Am J Infect Dis*, 2 (3), 187-192. PMCID: PMC1716153 PubMed: 17186064.
20. Turmelle, Y. P., Shikapwashya, O., Tu, S., Hruz, P. W., Yan, Q., Rudnick, D. A. (2006). Rosiglitazone inhibits mouse liver regeneration. *FASEB J*, 20 (14), 2609-11 PubMed: 17077279.

21. Hruz, P. W., Yan, Q. (2006). Tipranavir without ritonavir does not acutely induce peripheral insulin resistance in a rodent model. *J Acquir Immune Defic Syndr*, 43 (5), 624-5 PubMed: 17133213.
22. Hruz, P. W., Yan, Q., Struthers, H., Jay, P. Y. (2008). HIV protease inhibitors that block GLUT4 precipitate acute, decompensated heart failure in a mouse model of dilated cardiomyopathy. *FASEB J*, 22 (7), 2161-7 PubMed: 18256305.
23. Hruz, P. W. (2008). HIV protease inhibitors and insulin resistance: lessons from in-vitro, rodent and healthy human volunteer models. *Curr Opin HIV AIDS*, 3 (6), 660-5. PMCID: PMC2680222 PubMed: 19373039.
24. Flint, O. P., Noor, M. A., Hruz, P. W., Hylemon, P. B., Yarasheski, K., Kotler, D. P., Parker, R. A., Bellamine, A. (2009). The role of protease inhibitors in the pathogenesis of HIV-associated lipodystrophy: cellular mechanisms and clinical implications. *Toxicol Pathol*, 37 (1), 65-77. PMCID: PMC3170409 PubMed: 19171928.
25. Tu, P., Bhasin, S., Hruz, P. W., Herbst, K. L., Castellani, L. W., Hua, N., Hamilton, J. A., Guo, W. (2009). Genetic disruption of myostatin reduces the development of proatherogenic dyslipidemia and atherogenic lesions in Ldlr null mice. *Diabetes*, 58 (8), 1739-48. PMCID: PMC2712781 PubMed: 19509018.
26. Guo, W., Wong, S., Pudney, J., Jasuja, R., Hua, N., Jiang, L., Miller, A., Hruz, P. W., Hamilton, J. A., Bhasin, S. (2009). Acipimox, an inhibitor of lipolysis, attenuates atherogenesis in LDLR-null mice treated with HIV protease inhibitor ritonavir. *Arterioscler Thromb Vasc Biol*, 29 (12), 2028-32. PMCID: PMC2783673 PubMed: 19762785.
27. Vyas, A. K., Koster, J. C., Tzekov, A., Hruz, P. W. (2010). Effects of the HIV protease inhibitor ritonavir on GLUT4 knock-out mice. *J Biol Chem*, 285 (47), 36395-400. PMCID: PMC2978568 PubMed: 20864532.
28. Gazit, V., Weymann, A., Hartman, E., Finck, B. N., Hruz, P. W., Tzekov, A., Rudnick, D. A. (2010). Liver regeneration is impaired in lipodystrophic fatty liver dystrophy mice. *Hepatology*, 52 (6), 2109-17. PMCID: PMC2991544 PubMed: 20967828.
29. Hresko, R. C., Hruz, P. W. (2011). HIV protease inhibitors act as competitive inhibitors of the cytoplasmic glucose binding site of GLUTs with differing affinities for GLUT1 and GLUT4. *PLoS One*, 6 (9), e25237. PMCID: PMC3179492 PubMed: 21966466.
30. Vyas, A. K., Yang, K. C., Woo, D., Tzekov, A., Kovacs, A., Jay, P. Y., Hruz, P. W. (2011). Exenatide improves glucose homeostasis and prolongs survival in a murine model of dilated cardiomyopathy. *PLoS One*, 6 (2), e17178. PMCID: PMC3040766 PubMed: 21359201.
31. Hruz, P. W., Yan, Q., Tsai, L., Koster, J., Xu, L., Cihlar, T., Callebaut, C. (2011). GS-8374, a novel HIV protease inhibitor, does not alter glucose homeostasis in cultured adipocytes or in a healthy-rodent model system. *Antimicrob Agents Chemother*, 55 (4), 1377-82. PMCID: PMC3067185 PubMed: 21245443.
32. Hruz, P. W. (2011). Molecular mechanisms for insulin resistance in treated HIV-infection. *Best Pract Res Clin Endocrinol Metab*, 25 (3), 459-68. PMCID: PMC3115529 PubMed: 21663839.
33. Remedi, M. S., Agapova, S. E., Vyas, A. K., Hruz, P. W., Nichols, C. G. (2011). Acute sulfonylurea therapy at disease onset can cause permanent remission of KATP-induced diabetes. *Diabetes*, 60 (10), 2515-22. PMCID: PMC3178299

PubMed: 21813803.
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